ON THE PROBLEM OF CEREBELLAR VASCULAR LESION

P. Hubenov, L. Havezova, S. Geneva, A. Dimitrova

Key-words: cerebellar infarction — clinics — electrophysiology — pathomorphology

Cerebellar vascular lesion is most commonly detected only at autopsy. This fact determines to a great extent the actuality of the problem. Cerebellar haemorrhages are considered mainly in clinico-morphological aspect (1, 3, 5) rendering an account of the difficulties of making the clinical diagnosis, the variety of the clinical pattern, the dependence on the localization and size of cerebellar extravasate. N. V. Levedeva (1974) and Z. L. Luriya (1959) point out the dominant role of certain clinical symptoms. Other investigators (2,6, etc.) reveal the clinical picture of ischemic cerebellar lesion and the difficulties of its diagnosis.

The aim of the present work is to analyze some peculiarities of the clinical pattern as well as the electrophysiological and morphological alterations of cerebellar stroke.

Material and methods

During a 7-year period (from 1975 till 1982) a total of 34 patients with cerebellar vascular lesion were hospitalized in the Clinic of Neurology of the Higher Institute of Medicine, Varna. In any cases a pathologo-anatomic verification was done. There was a haemorrhage in 21 cases and an ischemic lesion in 13 ones. The patients were aged between 41 and 82 years with the higher incidence rate in the age group between 60 and 70 years (11 cases). There were 13 female and 8 male haemorrhage patients, and 6 female and 7 male ischemia ones.

Hypertension came first etiologically combined with atherosclerosis in 24 patients. Next was the generalized atherosclerosis: established in 6 patients; in other 3 cases a rheumatic vasculitis, an aneurysm and an acute infectious disease was found out, respectively.

Clinical course of cerebellar haemorrhages was rather various. It was notable that the disease had begun acutely in 16 cases manifested with occipital headache, vomiting, vertigo, psychomotor excitement or sleepiness followed by progressing hypotension on the background of which a discrete irritating pyramidal symptomatics persisted. A meningoradicular syndrome was observed in 12 cases; contracted pupils, anisocoria, swimming eye bulbs, and vision paresis — in 6 ones; hyporeflexia, nystagmus, disorders of respiration rhythm and frequency — in 10 ones. The patients died after one up to eleven days.

The illness began with immediate loss of consciousness, tonic and clonic convulsions followed by rapid lethal outcome with brain stem dysfunction phenomena in the early hours after the onset of incident in 4 patients. There was also a pseudotumour syndrome pattern with gradually slow increase of both focal and general brain symptoms determined by a haemorrhage in the left cerebellar hemisphere in one patient.

In any patients the paraclinical blood examination showed leukocytosis, the cerebrospinal fluid from the lumbar puncture was erythrochromic, and EEG indicated brain stem dysfunction without any focus of pathological activity in cerebral hemispheres.

Pathologoanatomically, haemorrhage was located in one cerebellar hemisphere without rupture into liquor spaces in one patient only; there was a large hematoma involving both hemispheres in 2 cases; with hematoma complicated with rupture into the subarachnoid space were other 2 patients; and with rupture into the IVth ventricle were 3 patients; simultaneously into it and the subarachnoidal space — in other 8 ones. Hemorrhage involved also the brain stem in 5 patients. Cerebellar tonsil inclinations presented a common feature.

In any patients the course of cerebellar infarction was accompanied by occipital headache, vomiting, cerebellar hemiataxia, dysarthria and muscular hypotonia. Acutely arising cerebellar symptoms were falsely considered a central hemiparesis or aphasia in 6 patients. In case of large ischemic foci brain-stem signs were also detected such as strabism, dysphagia (in 3 patients), as well as general brain disturbances with quantitative changes in consciousness clearness.

The paraclinical tests, lumbar puncture, EEG and X-ray examinations did not show any alterations to confirm the topics of the process. A bilateral carotid angiography provided data about an internal hydrocephaly in these 2 patients.

Pathologomorphologically: there was an infarction in one cerebellar hemisphere in 7 cases, a combination between cerebral and cerebellar ischemia in 2 ones and between cerebellar and brain stem one in 4 patients. Intracranial hypertension signs due to pressure of the cerebral aqueduct by oedematous and necrotic cerebellum were established, too. In 3 cases old cysts caused by previous disturbances of cerebral circulation in the same regions were observed.

The clinical diagnosis was cerebral hemisphere ischemia in 8 patients, space occupying process in 2 ones, and brain stem ischemia in 3 only.

On the basis of this study the following conclusions can be drawn:

1. The clinical diagnosis of cerebellar vascular lesion is rather difficult because focal symptoms are concealed by the clinical manifestations of brain stem compression and intracranial hypertension. However, for all that consciousness alteration, quadrihypotonia, discrete irritating bilateral pyramidal symptomatics and xanthochromic cerebrospinal fluid allows to suspect a cerebellar hemorrhage.

2. The gradual development of unilateral cerebellar symptomatics is an argument in favour of an ischemic cerebellar lesion.

3. With a view to the early diagnosis a complex of EEG and REG examination, vertebral angiography and computed axial tomography has to be more widely used.

REFERENCES

1. Антонов, Н., С. Божинов. Диференциална диагноза на неврологичните заболевания. С., Медицина и физкултура, 1980 с. 48. — 2. Марков, Д. А., Э. И. Злотник, Л. С. Гиткина. Инфаркт мозга. Минск, 1973, 174 с. — 3. Настев, Г. Мозъчен инсулт. С., Медицина и физкултура, 1972. — 4. Педаченко, Г. А., И. П. Пастушин. Кровоизлияния в мозжечок. Киев, Здоровья, 1975. — 5. Хаджиев, Д. Съдови заболявания на нервната система. С., Медицина и физкултура, 1979. — 6. Харалланов, Х. Недостатъчност на вертебро-базиларното кръвообращение. С., Медицина и физкултура, 1974.

К ВОПРОСУ О ВАСКУЛЯРНОЙ ЛЕЗИИ МОЗЖЕЧКА

П. Хубенов, Л. Хавезова, С. Генева, А. Димитрова

РЕЗЮМЕ

Авторы приводят результаты анатомо-клинического исследования 34 больных с васкулярной лезией мозжечка. Подчеркиваются трудности при диагностицировании. Устанавливается, что кровоизлияния мозжечка характеризуются преимущественно интенсивной головной болью, альтерацией сознания, квадрогипотонией, дискретной билатеральной ирритативной пирамидальной симптоматикой и кровавым ликвором, а при ишемической лезии мозжечка — постепенно развивающейся унилатеральной церебеллярной симптоматикой.